often been elevated in the few patients with reported values, but cortisol is elevated in many seriously ill patients who are not ketotic. Although ethanol causes structural hepatic mitochondrial abnormalities,[10] if those abnormalities cause ketosis, why should the ketosis disappear so quickly after glucose administration? The mitochondrial structural abnormalities presumably do not. In nearly all the reported cases the patients have severely curtailed their food intake, usually for at least several days. Such starvation, superimposed on chronic malnutrition, together with the ketogenic effect of ethanol,[11] may be important in the pathogenesis of the ketosis (and the hypoglycemia). If that were the whole story, however, why do we not see more alcohol abusers with these syndromes? Perhaps the abnormalities are more common than we realize, but disappear when patients stop drinking, if only long enough to eat sufficient carbohydrate.

Treating the basic disorder—chronic alcohol abuse—is not very successful. The treatment of the hypoglycemia and ketosis, however, is; both respond rapidly to administration of glucose. Patients who are dehydrated, as many are because of vomiting, should also receive parenteral sodium chloride solutions, which may also promote the renal excretion of β-hydroxybutyrate and acetoacetate. Some patients, especially those with severe vomiting, may also require administration of potassium salts after renal function has been shown to be adequate. Whether some patients should also receive phosphate, especially because starved alcoholics may be phosphate-depleted and profound hypophosphatemia may develop during treatment (although initially they may have *hyper*phosphatemia), has been discussed by Miller and colleagues.[3]

Alcoholic ketosis is defined as a syndrome occurring in nondiabetic persons. However, having diabetes mellitus surely does not protect a vomiting, starved chronic alcoholic from ketosis. Therefore, if a patient with presumed alcoholic ketosis is also diabetic—or if not known to be diabetic, develops severe hyperglycemia during treatment with glucose—it may be prudent to administer small doses of insulin to mitigate such hyperglycemia.

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Nationalized Health Care—A Juggernaut in 1981

WEBSTER DEFINES a juggernaut as "a massive inexorable force or object that crushes whatever is in its path." Some very recent events raise the possibility of a federal juggernaut for nationalized health care in 1981. If the person elected as President in 1980 rides the crest of an emotional wave of frustration with the present and of nostalgia for the past, and if that President were to have had a long experience in Congress and an essentially unchangeable personal commitment to nationalized health care, there could develop a situation much like that in 1965. A *landslide* presidential victory in 1964 permitted a legislatively skillful President to get a sweeping social program in health care enacted into law on a crash basis, with very little concern being given to its fiscal or other consequences. The consequences have now become familiar. There has been an unanticipated and frightening escalation in costs, in no small part due to clumsy and bungling attempts at planning, regulation and control of the health care enterprise. In all fairness the goal of more and better care for more people has been achieved but the real dollar cost of this achievement was neither calculated nor foreseen.

The possibility of a similar scenario for 1980 and 1981 seems real. The mood of the country appears at the moment to favor fiscal restraint and reducing the cost of government. Taxes, monetary inflation, unwanted dependence on foreign oil, and the rising price and uncertain availability of gasoline and other forms of energy are

all causing real concern. But there are also signs of smouldering frustration with these harsh realities and the growing restrictions they have begun to impose. One can sense that many people seem to be hoping, perhaps with a bit of nostalgia, for some sort of return or reincarnation of bygone times when money was more freely available and governmental spending seemed to solve any sort of problem. The young may especially resent the restrictions and discipline that are asked for, while the elderly may particularly resent the inflation and higher prices that erode their savings and carefully laid plans for retirement. Both are important voting blocks and they are joined by many other groups that stand to gain from government spending and further proliferation of government programs. This is highly combustible fuel for a health care juggernaut in 1981.

If nationalized health care is truly a good thing, then so be it. If it is not, then measures to counter or derail a possible juggernaut should begin soon. Most physicians and many others believe that nationalized health care is not in the long-term public interest and that there are better alternative solutions to the health care problems, which for whatever reasons seem to be so much a part of the public's agenda. There appear to be a number of basic points at issue:

- Is there a genuine need for nationalized health care? If so, what are the real or perceived problems in health care that are not now being met but which nationalized health care would solve? How could these needs be met if health care were not nationalized?

- The issue of health care costs is a major focus of attention. Would nationalized health care reduce or increase costs—and, if so, how? Can the existing health care enterprise or system control or reduce costs without nationalization—and, if so, how?

- Responsiveness is important in health care; that is, responsiveness to individual patient care needs and responsiveness to scientific and technologic advances in health care. Will nationalized health care be more or less responsive—and how? Can responsiveness in the existing health care enterprise be improved?

- Is autonomy and independence (freedom) in patient care important? And how important is autonomy and independence for the organizations and institutions involved in health care? What is to be done about the reality of *interdependence* among doctors, patients, and organizations and institutions involved in health care, as well as the role and responsibility of government which have developed so greatly since 1965?

It is suggested that the above are among the fundamental issues in health care to be addressed at this time.

This writer believes that the issue of autonomy and independence (call it freedom if you will) within an inescapable reality of de facto interdependence (which after all really calls for some kind of regulating or control mechanisms if an interdependent system is to work smoothly) is the core issue to be addressed. If some workable and acceptable accommodation can be found for this fundamental issue, it seems likely that the others will begin to fall into place. Basically the regulations or controls that are needed for any interdependent social system to work smoothly must be either external—that is, imposed from the outside—or internal—evolving within the system itself. The present proponents of nationalized health care appear to assume that the regulation and control will be carried out by the federal government through laws and bureaucratic rules, and therefore imposed from the outside. This approach by definition cannot help but restrict, if not destroy, freedom and independence for patients, physicians, and the many organizations and institutions within the health care system. But, given the interdependence that is now a fact of life, is there a better alternative? Perhaps there is. If there is, and if autonomy and independence are to be preserved for all concerned, then one must look within the system for voluntary collaboration and coordination (which can be accomplished without loss of autonomy or independence) to find acceptable and workable solutions to health care problems within the realities of the interdependence among the parts of the system.

If all this is indeed the case, as it seems to be, then it is time for the leadership of a still somewhat free and independent health care enterprise to study collaboratively each of the issues listed above, and no doubt others, and to develop a response or a plan from within the existing health care system to deal more efficiently and effectively with each of the problems that nationalized health care would be expected to solve. Perhaps some sort of summit conference of the national health care enterprise is needed as an initial step, to begin a collaborative effort to (1) identify and

define the problems which must be solved, (2) decide what needs to be done and (3) develop a strategy of collaboration and coordination to get it done. This is long overdue and should now be accomplished as soon as possible, before any sort of nationalized health care juggernaut begins to gather momentum in the nation. Actually there may be very little time to spare. The scenario may have already started.

—MSMW

Phenylketonuria and Its Variants

OF ALL THE 150 or 200 inborn errors of metabolism known today, phenylketonuria (PKU) is the best known and the most studied. The reasons for this are clear: PKU is one of the commonest of these diseases (one in about 14,000 live births in the United States, even commoner in some ethnic groups), if untreated it leads to lifelong severe mental retardation (more devastating to the average family than, say, death in infancy) and an effective treatment is readily available. It is this last feature that has led, over the last 30 years, to the great interest in PKU among pediatricians and has caused many states to introduce screening of all neonates, as described by Dr. Charles Parker elsewhere in this issue. We can control the clinical features of PKU better than those of most inborn errors of metabolism—it is paradoxical though rewarding that our very success in treating PKU will in future severely limit opportunities to study the natural history of the disease. On the purely scientific side there have also been advances, though these have been slower than in clinical management and screening.

The collaborative study of children treated for PKU, referred to by Dr. Parker, was originally set up to determine whether dietary treatment had any value. Although at the time some felt this to be supererogatory, much valuable information has been produced by the study and our knowledge of PKU has been materially advanced by it. In the last few years, the collaborative study has been concentrating on when the dietary treatment should be terminated. Even in the early 1950's, pediatricians hoped that a normal diet could be resumed after a few years and some were courageous enough (or were forced by circumstances) to try the experiment. Scattered conflicting reports of the effects of terminating treatment appeared in the literature and the collaborative study has started a systematic investigation.

The low-phenylalanine diet was introduced because it was hypothesized that the high concentration of phenylalanine in the blood damaged the brain from soon after birth onwards and, as often happens with brain damage in infancy, this showed itself as global mental retardation.[1] However, a similar insult to a more mature brain would be expected to produce a different spectrum of signs and symptoms; although there might well be intellectual deterioration—that is, dementia—this would probably be relatively slow, and a more sensitive indicator of late onset phenylalanine intoxication would be desirable. Intelligence quotient tests were first used in the early 1950's for following effects of dietary treatment[1] and have been widely used since. They are probably the best indicators we have of brain damage caused by hyperphenylalaninemia in early infancy; however, there are better tests for the brain damage that might result from late onset phenylalanine intoxication—for example, tests of attention span, the categories subtest of the Reitan-Halstead battery, and structured or semistructured psychiatric interviews.

The early literature reported a handful of cases of "atypical PKU": cases of persons with normal or near normal intelligence quotients and with substantially raised blood phenylalanine levels and urinary excretion of phenylpyruvic acid and other "abnormal" phenylalanine metabolites. With improved laboratory techniques it was shown that these persons had blood phenylalanine concentrations well below those of typical patients with PKU, though considerably above the normal. The frequency with which these cases occurred became apparent when screening of the newborn was introduced—about one in 30,000 live births or one in three of all those with hyperphenylalaninemia in the United States were affected. These cases were labeled hyperphenylalaninemia variants or *hyper-phe*. Phenylalanine hydroxylase is the enzyme that normally converts phenylalanine to tyrosine but is absent or inactive in persons with typical PKU; in those with variant forms of hyperphenylalaninemia there is appreciable phenylalanine hydroxylase activity in the liver, though far less than in the normal. In some of these persons there is evidence suggesting a qualitatively altered phenylalanine hydroxylase which in turn suggests a structural gene mutation,